

Regulation of K^+ Ions Concentration

The Normal concentration of K^+ ions = 3.5 Meq/dl.

The Average is 4.2 Meq/dl.

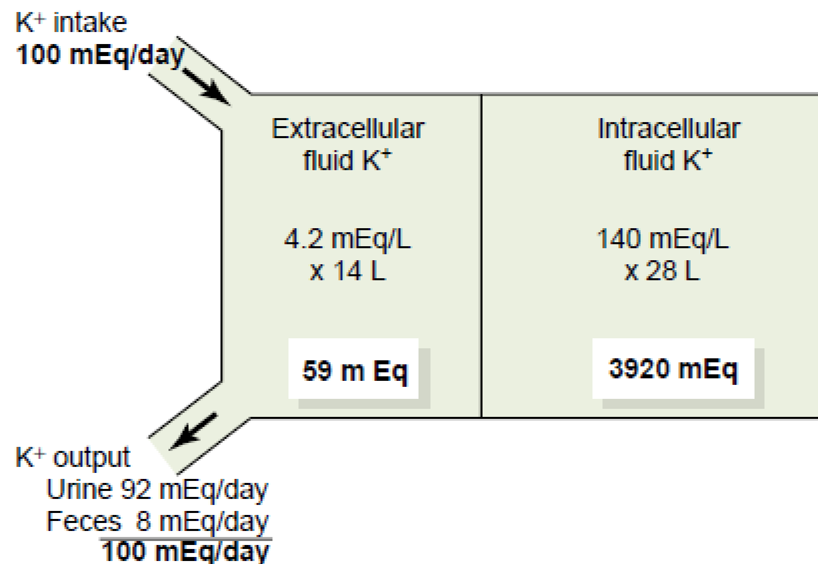


Figure 29–1

Normal potassium intake, distribution of potassium in the body fluids, and potassium output from the body.

Regulation of K^+ ions is very important because any change will make disease (problems) Hypokalemia (decrease K^+ concentration) will cause severe muscle weakness.

Hyperkalemia is more dangerous and cause many problems :

1. Cardiac arrhythmias.
2. Cardiac arrest.

Concentration of K^+ inside the cells is 140 Meq/dl higher than extracellular fluid

Excess K^+ in the body is mainly excreted by kidneys and small part of K^+ can be excreted by stool.

There is a first line of defense against change in K^+ concentration, which is redistribution of K^+ in extracellular fluid and intracellular fluid. For example if a person eats a meal rich with K^+ we say that K^+ concentration in this meal is 40 mq, after meal the K^+ will increase in extracellular fluid.

If we want to know the increase in K^+ concentration in extracellular fluid, we have to know the fluid amount in extracellular fluid, which is 14L
so 40/14 = 2.9 meq/L
So first line of defense is redistribution between extracellular fluid and intracellular fluid. Excess K^+ goes to the cells gradually.

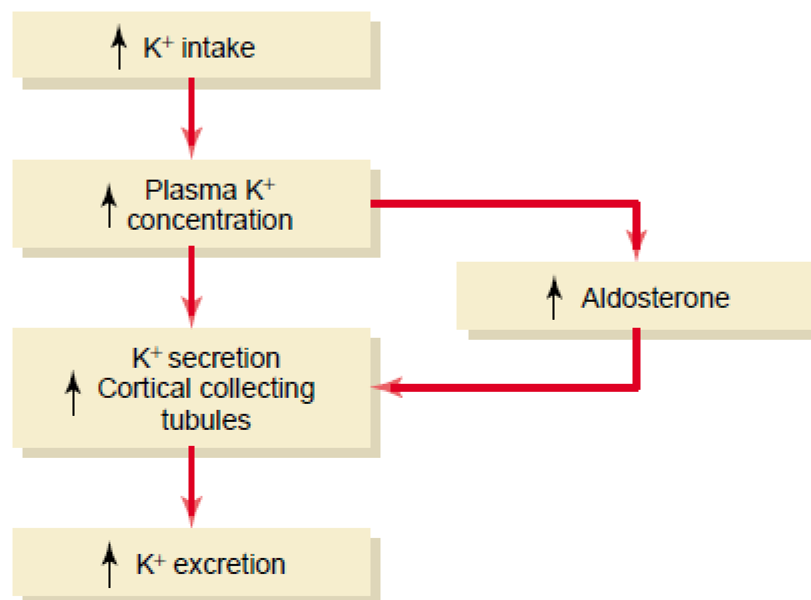


Figure 29–7

Primary mechanisms by which high potassium intake raises potassium excretion. Note that increased plasma potassium concentration directly raises potassium secretion by the cortical collecting tubules and indirectly increases potassium secretion by raising plasma aldosterone concentration.

Factors that affect redistribution:

1. Insulin hormone after meal: insulin is secreted and this increases the uptake of K^+ by cells. So diabetic after meal suffer of hyperkalemia.
2. Aldosterone hormone (secreted by renal cortex) increases the uptake of K^+ by cells.
3. Catecholamine (espically Epinephrin) increases the uptake of K^+ by cells by the activation of beta receptor cells.
   So people who takes drugs which inhibit the receptors (Propanolol) (Inderal) used for treatment of hypertension and other cardiovascular diseases, so people who take this will have tendency to severe hyperkalemia.
4. Cellular Lysis: causes hyperkalemia if massive destruction of cells occur like RBC or muscle injury.
5. In prolonged heavy exercise can occur mild hyperkalemia because skeletal muscle release K^+ during exercise. But patient with diabetes mellitus or who is treated with (Inderal) after prolonged exercise will suffer of significant hyperkalemia.

Table 29–1

Factors That Can Alter Potassium Distribution Between the Intra- and Extracellular Fluid

Factors That Shift K^+ into Cells (Decrease Extracellular [K^+])	Factors That Shift K^+ Out of Cells (Increase Extracellular [K^+])
• Insulin	• Insulin deficiency (diabetes mellitus)
• Aldosterone	• Aldosterone deficiency (Addison's disease)
• β-adrenergic stimulation	• β-adrenergic blockade
• Alkalosis	• Acidosis
	• Cell lysis
	• Strenuous exercise
	• Increased extracellular fluid osmolarity

Excretion of K+ by kidney:

Excretion by principle cells found in late distal and cortical collecting tubule, Na-K pump cause influx of K which increase concentration in cells, so concentration difference of K^+ between cells and lumen occur → diffusion of K^+ to lumen.
N.B. tubular membrane is permeable to K^+.

Other cells in this site called intercalated cells, in severe hypokalemia reabsorption occur by these cells but excretion does not occur (by primary active transport).

Factors that affect excretion of K by principle cells:

1. K concentration is high: this increase excretion by increasing the activity of Na-K pump and stimulate the secretion of aldosterone by adrenal cortex, this hormone increase excretion by increasing activity of the pump and increasing the permeability of the membrane.
   - when aldosterone is low, hyperkalemia occur → Addison's disease
   - when aldosterone is high, hypokalemia occur → Con's syndrome
2. Increase distal tubular flow rate. When diuretics are used, the flow of fluid increase in distal tubules and excretion of K also increase because concentration difference between cell and lumen is high because the flow of fluid is high, so in this case concentration of K is minimal, so difference in concentration increase and excretion of K^+ increase.
3. Acute and chronic acidosis:
   - Acute acidosis: K^+ excretion decrease because Na-K pump decrease in its activity.
   - Chronic acidosis: K^+ excretion increase because of chronic inhibition of reabsorption of Na-Cl-Water in proximal tubule so increase distal tubular flow rate and increase in K^+ excretion.

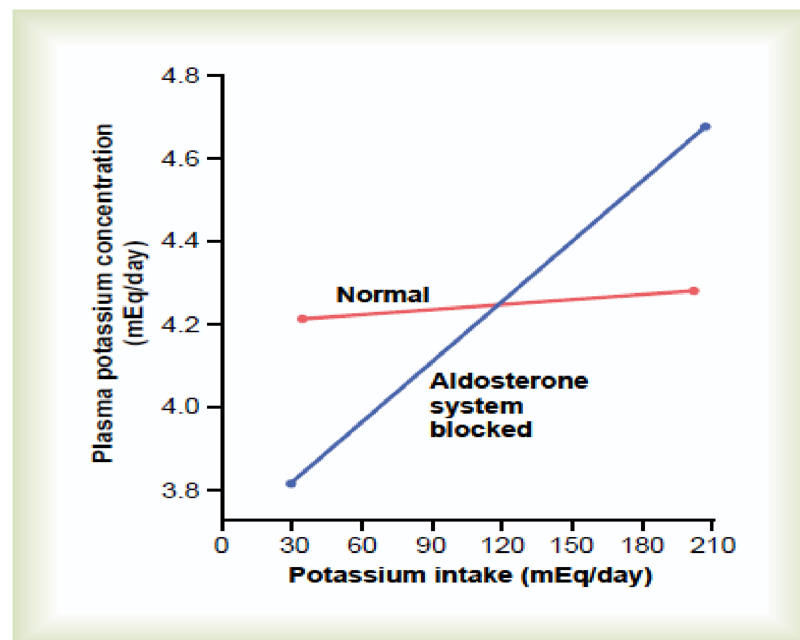


Figure 29–8

Effect of large changes in potassium intake on extracellular fluid potassium concentration under normal conditions (*red line*) and after the aldosterone feedback had been blocked (*blue line*). Note that after blockade of the aldosterone system, regulation of potassium concentration was greatly impaired. (Courtesy Dr. David B. Young.)

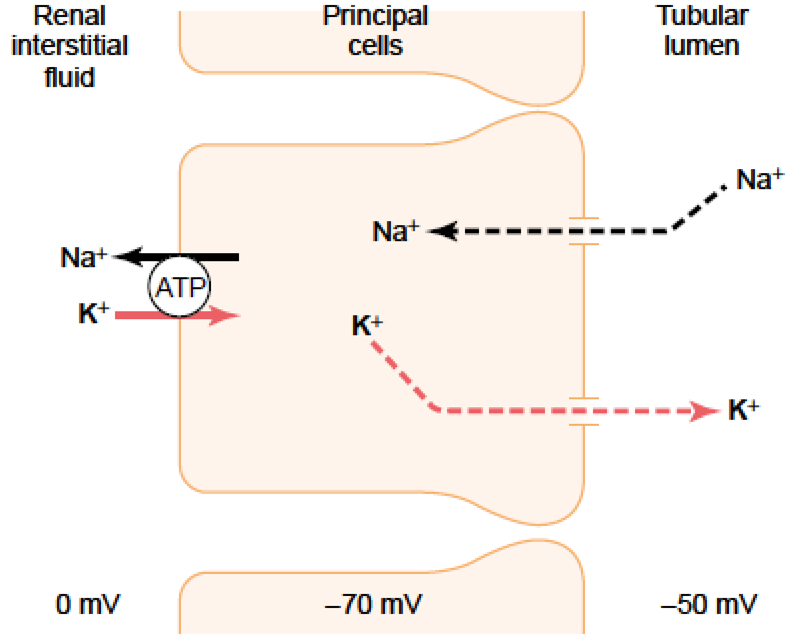


Figure 29–3

Mechanisms of potassium secretion and sodium reabsorption by the principal cells of the late distal and collecting tubules.

هذه الملخصات هي منكم. عن اجتهاد شخصي لمجموعه من طلاب الطب البشري /عدن معتمدين على عدة مصادر مما تم محاضرته او قراءته ..وقد تفيد البعض منكم .وللعلم ليس لها اية علاقة مباشره بأي عضو من اعضاء الهيئة التعليمية في الكلية. *زملائنا الأعزاء إن أصبنا فمن الله ، و إن أخطأنا فمن أنفسنا و من الشيطان " نبقى بشرا نخطئ و نصيب فلذا يرجى الإشعار في حالة وجود اي ملاحظات عبر ايميل الجروب

Sharencare@outlook.com